Amendments to the Specification:

Insert the paper copy of the Sequence Listing filed herewith following the Abstract.

Please amend the paragraph beginning at page 23, line1 9, as follows:

Human embryonic kidney 293 cells were seeded into 48-well culture plates at 105 cells per well in DMEM supplemented with 10% fetal bovine serum. After 24 hours, cells were transfected by a calcium phosphate coprecipitation method with 250 ng of the pGL3/UREluc reporter gene which consists of three copies of AGGTCAagccAGGTCA (SEQ ID NO:12) fused to nucleotides -56 to +109 of the human c-fos promoter in front of the firefly luciferase gene in the plasmid basic pGL3 (Promega), 40 ng pSG5/hRXRa, 40 ng pSG5/rUR or CMX/hLXR, 10 ng pSG5/hGrip1, 0.4 ng CMV/R-luc (transfection normalization reporter, Promega) and 250 ng carrier DNA per well. Alternatively, 500 ng of the pGL2/7aluc reporter gene which consists of a single copy of nucleotides -101 to -49 of the rat 7a-hydroxylase gene fused to the SV40 promoter in front of the firefly luciferase gene in the plasmid basic pGL2 (Promega) was used instead of pGL3/UREluc. This reporter does not have response elements for COUP-TFII or HNF4. In some experiments, 500 ng of the human 7α-hydroxylase gene reporter, PH/hCYP7A-135, which consists of a single copy of nucleotides -135 to +24 of the human CYP7A gene fused to the firefly luciferase gene in the plamid basic pGL3 (Promega), was used instead of pGL2/7aluc. After another 12-24 hours, cells were washed with PBS and refed with DMEM supplemented with 4% delipidated fetal bovine serum. Steroid derivatives dissolved in ethanol were added in duplicate to the medium so that the final concentration of alcohol was 0.2%. After 24-48 hours, cells were harvested and luciferase activity was measured with a commercial kit (Promega Dual luciferase II) on a Monolight luminometer (Beckton Dickenson). Both LXR and UR form heterodimers with RXR for gene transactivation. The ligand for RXR, 9-cis retinoic acid, is known to activate the LXR/RXR heterodimer but addition of 9-cis retinoic acid to transactivation assays did not change the potency of either Δ^5 or 6α-hydroxy steroids for activation of LXR or UR (data not shown). The expression of endogenous LXR and UR (and TR which also binds to

a DR4 response element) were apparently low since reporter activation was low in the absence of added expression vectors for LXR or UR. Reporter activation was also low when the DR4 response-element was replaced with a glucocorticoid receptor response element. Each experiment was repeated as least twice to demonstrate reproducability. Relative light units were about 2×10^7 for pGL3/UREluc, 1×10^6 for pGL2/7aluc, 5×10^4 for PH/hCYP7A-135 and 5×10^5 for CMV/R-luc. Purity of synthesized steroid derivatives was verified by thin layer chromatography and structures were confirmed using proton and C^{13} magnetic resonance spectrometry. 3-Oxo-6α-hydroxy-5β-cholanoic acid methyl ester, 3α,6α-dihydroxy-5β-cholanoic acid methyl ester, and 3α,6α,7α-trihydroxy-5β-cholanoic acid methyl ester were found to be as potent as 3β-hydroxy-Δ^5-cholanoic acid methyl ester as activators for LXR, with ED_{50}'s of about 150 nM. Loss of activity was seen when the 6α-hydroxy group was changed to a 6β configuration. In contrast to activity with LXR, 3β-hydroxy-Δ^5- cholanoic acid methyl ester (ED_{50} of 130 nM) was more active than 3-oxo-6α-hydroxy- cholanoic acid methyl ester (ED_{50} of 550 nM) and 3α,6α-dihydroxy-cholanoic acid methyl ester (ED_{50} of 500 nM) for UR activation.